

Abstracts of the 30th Annual Conference on Cardiovascular Disease Epidemiology

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**Third-Day Program
Workshop on Obesity and Blood Pressure
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**Cosponsored by the Council on Epidemiology of the American Heart Association and
National Heart, Lung, and Blood Institute**

Information about registration and hotel accommodations is available from the Conference Coordinator, Scientific and Corporate Meetings, American Heart Association, 7320 Greenville Avenue, Dallas, TX 75231. Telephone: 214/706-1511.

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Children's HDL-cholesterol: The Effects of Tobacco Smoking, Smokeless and Parental Smoking	33
Paul Poirier, Janet Hollarbush, William Clarke, Ronald Lauer. University of Iowa, Iowa City, IA	
Two projects, the Muscatine Ponderosity Study and the Prevention of Adolescent Smoking Project, provided an opportunity to relate tobacco exposure, particularly passive exposure due to parental smoking, and HDL-cholesterol levels. This report is based on the data gathered from the 431 students in grades 6 & 7 in the Muscatine, IA school system who participated in both surveys.	
Children whose parents smoke daily had lower mean HDL-cholesterol than those whose parents do not smoke: 49.0 mg/dl (n=209) vs. 51.7 mg/dl (n=242), $p<0.01$. This association remained significant independent of body mass index, age and gender.	
Regular tobacco use was reported by a small portion of students. Students smoking monthly or more have lower HDL-cholesterol levels (48.5 mg/dl (n=51) vs 50.7 mg/dl (n=399)). Boys who chew tobacco have lower HDL-cholesterol levels (47.1 mg/dl (n=27) vs 50.6 mg/dl (n=224), $p=.08$). In boys, but not girls, the combination of parental smoking and the child's chewing or smoking had the lowest HDL-cholesterol levels.	
These results suggest that tobacco exposure by personal use of cigarettes or smokeless and passive exposure all contribute to lower HDL-cholesterol levels in children.	

Change in Alcohol Consumption and Risk of All-Cause and Ischemic Heart Disease Mortality in the Alameda County Study

Nancy B. Lazarus, George A. Kaplan, Richard D. Cohen, Ding-Jen Lee. California Public Health Foundation, Berkeley, CA

Previous evidence suggests that those who abstain from alcohol consumption are at higher risk of ischemic heart disease (IHD) mortality. It remains controversial whether the increased risk is found among all abstainers or only those who recently quit drinking. Differentiating between long-term abstainers and more recent nondrinkers, the proportional hazards model was used to study the change in alcohol consumption from 1965 to 1974 and 10-year (1974-1984) all-cause and IHD mortality in 4,070 persons aged 35 and over. Women who were moderate drinkers who quit compared to light drinkers at both times are at increased risk of mortality from all-causes ($RH = 6.56$; 95%CI = 2.41 to 17.84) and from IHD ($RH = 10.49$; 95%CI = 2.50 to 44.01). This is true for men for all-cause mortality ($RH = 1.78$; 95%CI = 1.12 to 2.83) but not for IHD mortality ($RH = 0.75$; 95%CI = 0.31 to 1.83). For long-term abstainers compared to light drinkers, only men were at marginally increased risk and only for all-cause mortality ($RH = 1.26$; 95%CI = 0.91 to 1.75). Some of the increased risk associated with abstention appears to be due to a higher risk among those who quit.

Mortality in Exdrinkers	34
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Higher mortality in exdrinkers (vs. lifelong abstainers) has usually been attributed to a higher prevalence of illness among exdrinkers. Age-adjusted data for members of a prepaid health plan showed that 3810 exdrinkers reported more illness than 15,510 abstainers (47.0% vs. 36.1%) but cardiovascular (CV) risk was similar (40.3% of exdrinkers vs. 41.1% of abstainers). During follow-up from 1978-1985 (median follow-up = 5 years), 407 abstainers and 178 exdrinkers died. Age-adjusted Cox regression analyses showed that, compared to lifelong abstainers, exdrinkers had higher mortality from CV causes (relative risk [RR] = 1.5, 95% CI = 1.3-2.1, $p<0.01$) and from non-CV causes (RR=1.7, CI=1.3-2.1, $p<0.001$). Adjustment for additional covariates (sex, race, smoking, body mass index, marital status, education) reduced the mortality risk of exdrinkers: RR for CV = 1.0, CI=0.8-1.3 and RR for non-CV = 1.3, CI=1.0-1.7, $p<0.05$. Exdrinkers who stopped for medical reasons had higher mortality from non-CV causes (RR=1.5, CI=1.1-2.1, $p<0.01$), but not from CV causes (RR=1.1, CI=0.7-1.6). From these data, we conclude that higher non-CV mortality among exdrinkers is due substantially to baseline illness, but higher CV mortality among exdrinkers is due to confounding by other traits related to past alcohol use.	

CHD Risk Associated with Regular and Acute Consumption of Alcohol	36
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The association of regular and acute alcohol consumption with non-fatal myocardial infarction and coronary death was investigated in a case control study in New Zealand. A total of 1367 people aged 35-64 years without prior known coronary disease were included in the study. Interviews were conducted with 227 male and 72 female myocardial infarction cases identified in a community based MONICA register, and 525 male and 341 female controls, randomly selected from same population and group matched by age and sex. Data on coronary death cases (128 men and 30 women) identified in the MONICA register came from a close friend or relative. Similar information was obtained from a close friend or relative of 330 male and 214 female age and sex matched controls.	

There was a strong, consistent inverse association between alcohol consumption and both fatal and non-fatal CHD risk in men and women. After controlling for possible confounding, people who drank alcohol more than once per month had a 30-60% reduction in CHD risk. Ex-drinkers had a reduced risk of non-fatal myocardial infarction but not of coronary death. Alcohol consumption was also associated with an acute reduction in CHD risk. Among people who drank at least once per month, CHD risk was halved during the 24 hours after a drinking episode.

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